and glycol ethers. To the best of our knowledge, so far there has been only one case report of poisoning resulting from the consumption of brake fluid.[2]

After ingestion of ethylene glycol, inebriation occurs but the typical smell of alcohol is lacking. Ethylene glycol is metabolised by the enzyme alcohol dehydrogenase to glycoaldehyde, glycolic acid and glyoxylic acid, which are responsible for most of the clinical effects of ethylene glycol poisoning.[2] Cardiovascular toxicity of ethylene glycol usually appears after a period of 12–24 hours and is characterised by tachycardia, hypertension, and pulmonary oedema.[2] In all the three cases, apart from nausea and vomiting, the other clinical features were not present. Acute renal failure usually occurs as a delayed manifestation after 24 hours of ingestion in 73%–84% of cases of ethylene glycol poisoning [2] and was present in all of our cases. The presence of proteinuria in all cases was consistent with acute tubular necrosis and was confirmed on biopsy of the kidney in case number 1. All of our cases also had microscopic haematuria.

For patients presenting early with ethylene glycol poisoning treatment with ethanol is preferred. Ethanol acts by competing with ethylene glycol for the enzyme alcohol dehydrogenase thus limiting the formation of toxic metabolites. All three patients had presented late (>24–72 hours) with established renal failure. As the elimination half life of ethylene glycol is three hours [2] and more than five times the elimination half life had elapsed, there would have been little ethylene glycol left in the body. Hence, treatment with ethanol would not have served any therapeutic purpose. The modality of treatment chosen was haemodialysis to facilitate removal of toxic metabolites of ethylene glycol and to combat uraemia.

Besides ethylene glycol, the other components of brake fluid may also have played some part in the manifestations of this unusual poisoning. Diethylene glycol has been incriminated earlier in a case report of five cases of acute renal failure complicating the use of diethylene glycol based silver sulfadiazene ointment.[3] Recently, diethylene glycol has been implicated as the causative factor for renal failure in the paediatric population of Bangladesh, Haiti, and India.[4–6] The mode of poisoning was from contamination of the available liquid peadiatric medications with diethylene glycol. In another case report, propylene glycol has also been suspected of having led to hyperosmolarity and cardiorespiratory arrest in an infant.[7] All toxic glycols are metabolised by alcohol dehydrogenase resulting in profound metabolic acidosis attributable to the accumulation of organic acids. In all our cases, it is possible that the consumption of ethanol probably led to partial saturation of the enzyme alcohol dehydrogenase thereby limiting the formation of organic acids. The resultant metabolic acidosis was mild in all cases and significant toxicity on systems other than the kidneys was avoided.

Contributors

Navneet Sharma was the treating physician who diagnosed, managed and treated the patients in the medical emergency. Sanjay Jain, a senior consultant in the emergency department, actively participated in the discussion of treatment modalities and review of this paper. Navneet Sharma is the guarantor of the paper.

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Metal fume fever: a case report and review of the literature

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Metal fume fever is an acutely noxious inhalation syndrome secondary to metal oxide fumes. Despite preventative strategies sporadic cases are likely to continue to present to emergency departments.

A 55 year old man presented to the emergency department at 9 pm. He complained of feeling generally unwell for the previous five hours. He described malaise, fatigue, cough, fever, nausea, and dyspnoea at rest. He had no previous medical history of note and was usually fit and well. He was a non-smoker. He worked as a plumber and on the day of admission had been using an oxyacetylene torch to remove a steel tank.

On examination, he was unable to talk in full sentences. His respiratory rate was 24/min with an oxygen saturation of 94% in room air. Chest examination was normal. His pulse rate was 100/min and he was feverish at 39°C. There were no other findings of note. Blood gas analysis demonstrated acute type I respiratory failure with an arterial oxygen partial pressure of 8.8 kPa. There was a neutrophil leucocytosis but no other abnormality of baseline pathology. A chest radiograph revealed patchy opacification in the right perihilar area.

His 18 year old son, who had been working with him all day, presented to the emergency department simultaneously. He complained of malaise, nausea, vomiting, and cough. He had no previous medical history. Examination and investigation were unremarkable.

A further coworker presented to a local minor injury unit the same night with similar symptoms to the 18 year old patient. He required no medical intervention.

A diagnosis of metal fume fever was made and the 55 year old man admitted for observation and oxygen therapy. His son was discharged. By the following morning both had made a full recovery.

DISCUSSION

Metal fume fever (MFF)—"brass founders ague", "zinc shakes", "Monday morning fever"—is a self limiting inhalation fever attributed to a number of metal oxide fumes. The history is characterised by fever, headache, myalgia, fatigue, and dyspnoea. Other features include cough, thirst, a metallic taste, salivation, and a neutrophil leucocytosis. Radiography may demonstrate bilateral diffuse infiltrative pulmonary lesions. Pulmonary function tests demonstrate a significant reduction in vital capacity, transfer factor and arterial oxygen partial pressure. Urine and plasma metal levels may be increased.

Onset is typically rapid, occurring between three and 10 hours after exposure. Spontaneous recovery occurs within 24 hours. No long term complications are known.[1]

MFF is classically associated with zinc oxide fume exposure from welding galvanised steel or brass. It is also seen in association with high temperature zinc coating processes and metal pouring in brass foundries. Magnesium and copper oxide fumes are more rarely the causative agents. Approximately 2000 cases are reported annually in the United States.[2 3]

The pathophysiology is unclear but seems to reflect a direct toxic effect. The lack of a latent period and the fact that large proportions of a single workforce can be affected are against an immunological basis for the disease. [4] There is evidence of an exposure dependent neutrophil alveolitis in association with tumour necrosis factor α, interleukin 6, and interleukin 8 cytokine release from pulmonary cells.[4] Interestingly there is evidence of rapid adaptation after repeated exposure though the transient nature of this tolerance is reflected in the synonym "Monday morning fever". It has been postulated that tolerance occurs because of induction of metallothionein protein synthesis. These proteins bind to heavy metals preventing toxic metal accumulation. [5]

Diagnosis is based on clinical suspicion, clinical findings, and rapid resolution. Evidence of possible exposure is critical. Treatment is symptomatic. The syndrome needs to be differentiated from serious MFF seen after military smoke exposure, which typically has a biphasic response with severe relapse 24 to 48 hours after initial remission.[6] It should also be differentiated from true chemical pneumonitis after metal fume exposure. This is particularly associated with cadmium fumes but also occurs with manganese, mercury, and nickel. In the early stages, it may be indistinguishable from MFF but the pneumonitis is progressive and usually complicated by non-cardiogenic pulmonary oedema. Cadmium also injures the renal tubules resulting in acute renal dysfunction.[1]

Preventative strategies for MFF are aimed at reducing fume exposure concentrations.[7] "Toxic" levels have not been established. However, Fine *et al* have demonstrated that inhalation of traditionally safe levels of zinc oxide can produce MFF symptoms and a rise in plasma interleukin 6.[8] In the UK, the Reporting of Injuries, Diseases and Dangerous Occurrence Regulations (1995) place responsibility on employers to report MFF to the Health and Safety Executive once it has been diagnosed in writing by a doctor.[7]

CONCLUSION

MFF is a common, acute, severe occupational syndrome. Despite preventative strategies, sporadic cases continue to present to emergency departments. Recognition of the possibility of an inhalation syndrome requires an understanding of the aetiology and an adequate occupational history. Early recognition can prompt a more directed management approach and permit the exclusion of more serious inhalational syndromes.

Contributors

PK and HY were responsible for the diagnosis and management of the case, reviewed the literature and wrote the paper. IOS reviewed and advised on the paper and is the guarantor for the paper.

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